

solidify the originally fluid mass and split it up in a longitudinal or transverse direction."

It will probably be a long time before an adequate comprehension of the mechanism of muscular contraction is attained, and it is not to be expected that the subject could be completely elucidated within the limits of a single paper. I have simply pointed out the explanation which, after a consideration of the various facts, would seem to be the most likely one.

OBSTRUCTION OF THE CENTRAL RETINAL ARTERY.¹

REPORT OF A CASE.

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THE question of embolism versus thrombosis of the central retinal artery has for a great many years been discussed and argued until it might be said to be almost threadbare; and, curiously enough, many of the best observers have titled their cases embolism, while declaring their belief in probable thrombosis. That this question is still an unsettled one in medicine is sufficient apology for further contributions.

The following case I believe to be one of lateral thrombosis of the central artery of the retina. The evidence to be adduced, though not conclusive, strongly favors such a diagnosis.

F. W., aged seventeen years, twister in a mill, came to the Eye Clinic of the German Hospital November 18, 1903, with history as follows: One week ago, while returning home from a matinee, she suffered a sudden attack of partial blindness in the left eye. This was described as everything becoming black before O. S., except in the lower field, where vision remained. Subjectively she noticed that the top of the head of a person looked at was not seen. The condition did not improve, but, on the contrary, she thinks became worse in the succeeding few days. The attack was neither preceded, accompanied, nor followed by vertigo, headache, or any physical discomfort whatever. An interesting event in the history of the attack which must be considered, though it is, perhaps, a little sensational, is the following: The play which she witnessed was an extravagant melodrama, in which the heroine, during the third act, became suddenly blind, and continued so until the end of the play, with dramatic effect. The patient was much impressed, and, as she expressed it, could not get the afflicted heroine out of her mind. While thinking of this accident, she had proceeded but three squares from the theatre when her own trouble commenced.

¹ Read before the Ophthalmological Section, College of Physicians, December 15, 1908.

Her family history is good. Her father and mother are living and well. She is the third of eight children, the oldest twenty-one years, the youngest three years; all living and healthy, except the seventh, who died in infancy. Her personal history is good. She had scarlet fever, measles, and whooping-cough in childhood; she has never had rheumatism, has no intestinal nor digestive disturbance; shows no evidence of syphilis, congenital or acquired. She menstruated at thirteen and has done so normally since. Two years ago she had typhoid fever, and was probably quite ill. At the present time she is well developed, of good color, and apparently well balanced. She seems to be in no way neurotic. She experiences some shortness of breath after sudden exertion, such as running up stairs.

Dr. Henry F. Page examined her heart November 27th; in his report he states: "Patient well nourished, color good, mucous membranes normal in appearance. Radial pulse soft, receding, and slow. Pulse rate 63. Inspection of chest shows slight bulging of left side, due probably to a slight degree of lateral curvature. Apex-beat not visible, but determined normal by palpation and auscultation. Slight pulsation of vessels of the neck. Fine thrill felt over carotids. Soft, blowing, systolic murmur heard at aortic and pulmonary orifices transmitted to the carotids." As a result of his examination, he believes the murmur present to be functional in character.

A complete analysis of the urine was made in the chemical laboratory of the Medical Department, University of Pennsylvania, by Dr. John M. Swan. It is not necessary to read the figures; suffice it to say that the patient has nephritis, and is eliminating much less nitrogen, sulphates, and phosphates than normal. The albumin is less than 0.025 per cent. by Esbach's method; the casts are hyaline and very few in number. There is no sugar.

Upon admission, both eyes, conjunctiva slightly injected, pupils equal, irides freely active to light. Movements of eyeballs free in all directions. Central vision: O. S. (affected eye), 5/7.5. V. O. D., 5/5.

Ophthalmoscopic Examination. O. S., media clear disk margins everywhere blurred, but more so below. There is a diffused area of ischaemic infarction, mostly below, but especially marked in a localized patch about the size of the disk, situated 1 d. d. down and out. Into this patch runs an obliterated vessel from which has been a small extravasation. The lower temporal artery from its exit to a turn 1 d. d. from the disk is reduced to a thread; from there on it is of good size. A small branch from this artery running through the infarcted area is filiform, but peripherally recovers a normal diameter. Directly off the disk below are three small, indistinct, flame-shaped hemorrhages. Most of the arteries and veins are reduced in size; they all taper as they enter the disk, and many of them show a most irregular contour varying with diameters. The light streak on the smaller vessels is absent, and on the larger is diminished in

brilliancy, but continued far to the periphery. The light streak on a few of the upper ones is beaded. The vessels are nowhere tortuous, and peripherally they seem normal. The macular region shows a number of reflexes, streaked and radiating. The appearance here is granular, but there is no cherry-red spot. The porus opticus is enveloped in an impenetrable mist. Pressure on the eyeball causes a complete blanching of the disk, with not a vessel on the disk to be seen.

There is a little variation from the usual arrangement of the vessels. The majority of them, for instance, seem to come from the superior branch of the first bifurcation, there being at least seven from this source against three or four from the inferior branch. Among those

FIG. 1.



Obstruction of the central retinal artery. O. S., probably lateral thrombus.

first mentioned is a large artery running directly outward and curving above the macula, in close proximity thereto. This vessel, although it points to the lower retinal artery, probably belongs to the upper system because it passes above the macula and supplies the upper outer fundus.

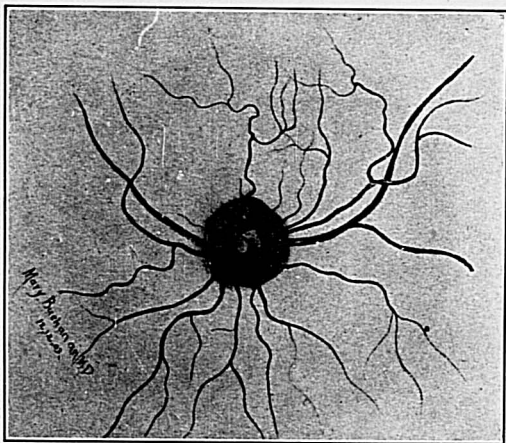
O. D. is normal ophthalmoscopically, but shows the same arrangement of the vessels. The upper fundus is much more vascular than the lower, and there is also a large macular artery, but in this eye it is a branch of the inferior system. There is in both eyes a tendency to lateral distribution of the vessels.

Dr. Buchanan, who has followed this case with me from the start, and who has been of great assistance in its preparation, has made

the water-color sketch, which shows well the condition about ten days after the attack. The rough sketch of O.D. shows the arrangement of the vessels in that eye.

Subsequent changes noted at intervals of two or three days have all been in the direction of increasing haziness of the retina (secondary oedema), enlargement of the veins and some increase in the diameter of the arteries. The small hemorrhages have disappeared. The infarcted area has become less pronounced by contrast. Vision gradually reduced until seven days after the first note it was 5/15. Since then it has improved, and at the present time it is 5/10.

FIG. 2.



Vascular arrangement of fundus. O. D.

November 25th. Two weeks after the attack a punctate deposit appeared on the posterior surface of the cornea, rather general in distribution, with a tendency to conical formation. This deposit has almost entirely disappeared. Fine vitreous opacities are now present. There has been no alteration in tension. The fields correspond to the fundus condition.

The clinical signs of thrombosis as given by Priestley Smith, in 1884 (*Ophthalmic Review*), and which might be said to have become classic, are:

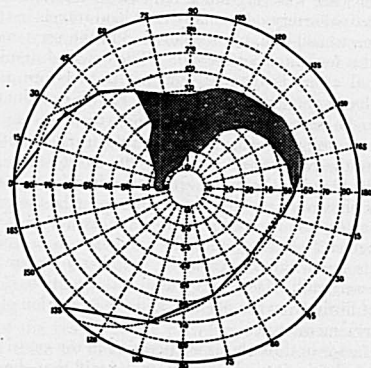
- (a) Previous attacks of transient blindness in the affected eye.
- (b) Simultaneous attacks of transient blindness in the fellow-eye.

(c) Previous or subsequent attacks of transient blindness in the fellow-eye, especially if conditions of the attack were the same in the permanent as in the transient attacks.

(d) Signs of disturbance of the cerebral circulation at the onset of the blindness—dizziness, faintness, headache.

In the history of the present case there are no points of correspondence with the above, but investigation along other lines would seem to make questionable the necessity for any one of these signs in establishing a case of thrombosis. It is true that with these clinical signs the case is probably thrombosis, but, with any or all absent, thrombosis is not to be excluded. In other words, when positive they are strong, and when negative they are weak. This case has been

FIG. 3.



Field. O. S.

studied therefore more from the side of general medicine and general pathology than from the clinical.

There are three important causative factors in the coagulation of blood within the vessel wall during life, two or all of which are for the most part essential.¹ These factors are alteration in the blood current, changes in the vessel wall, and alteration in the blood.

Ludwig Gutschy, in a recent article in the *Beiträge* for pathological anatomy and general pathology, has arrived at conclusions as to thrombus formation which seem to be of service in considering cases such as the one here reported. The first change he declares is one of separation; the fibrin separates from the blood plasma, forming a jelly-like mass, which adheres to the vessel wall, and

¹ Stengel. Text-book of Pathology.

which he calls the *primary fibrin membrane*. Upon this membrane, which is sticky, leukocytes collect, the resulting mass being a white thrombus. T. Wharton Jones¹ has found that on pressing the neck over the artery or vein firmly with a blunt point, an agglomeration of colorless corpuscles, with a few red ones, held together apparently by coagulated fibrin, occurs, adheres to the wall of the vessel, and more or less completely obstructs it at that place.

Returning now to the three factors more or less essential for thrombosis; under the first, or alteration in the blood current, we have to consider anything which slows the blood current, such as weakness of the heart, narrowing of the bloodvessels, or pressure on the vessel wall. I think it can be demonstrated that the blood current in this case was very much reduced in velocity, especially in the central retinal artery. Normally the blood travels in the carotids from 300 mm. to 500 mm. per second, and in the very small arteries it travels but a few mm. per second.² The external diameter of the central retinal artery is about $\frac{1}{10}$ mm.; it is therefore a very small artery, and has a very small lumen. Dr. Page's examination shows the pulse to be soft, receding, and slow, with a rate of 63 per minute. His examination was made under the usual conditions which tend to excite the patient and accelerate the heart-beat. If now the velocity of the blood in the radial artery is reduced below the normal to the extent shown by a pulse of 63, what must be the reduction in the central artery of the retina if we take as a standard Foster's figures just given? Furthermore, the axial stream in which the red cells travel is much quicker than the peripheral stream of plasma next the vessel wall. Here is certainly a condition favoring the separation of fibrin from the plasma, and the formation of Gutschy's primary fibrin membrane.

The next factor in thrombosis is alteration in the vessel wall. The patient has nephritis; it might, therefore, be said that she has endarteritis, arteriosclerosis, etc., but it is not likely, however, much as it would simplify things, if she had. Some change in the bloodvessel wall is very desirable for the purposes of this paper. This want is to be supplied by the bifurcation of the vessel. The point of bifurcation of the central artery of the retina—that point projecting in mid-stream, as it were—must necessarily stand more opposed to the onward flow of blood than the unobstructed wall. Such a mechanical arrangement causes a sudden halting of the stream with consequent embarrassment of some of the elements. Under these circumstances, and with an abnormally slowed current, the above-mentioned changes of separation might well take place. This might also be favored by some individual peculiarity in the angle and form of bifurcation. This case shows peculiarities in the vessels of the

¹ Guy's Hospital Reports, series 2, vol. vii. pt. 1.

² Foster's Physiology.

fundus, and it is not unreasonable to think that the vessels are also peculiar farther back. In support of this explanation is the experiment of Wharton Jones.

Changes in the blood itself have not been found. Two analyses were made by Dr. George P. Mueller, the second including an estimation of the fibrin. The first count gave: hæmoglobin, 100 per cent.; red cells, 4,650,000; white cells, 7650. Second count: hæmoglobin, 80 per cent.; red cells, 4,430,000; white cells, 5800. No increase in fibrin. Differential, normal.

This was somewhat of a disappointment as a functional heart murmur, which this undoubtedly was, is not usual with normal blood. My brother, Dr. Harvey Shoemaker, suggested that the blood quantity of the patient might be reduced as a whole. Cabot is authority for the existence of this condition. He has found it in typhoid fever, and attributes it to a concentration of the blood. There is no concentration of blood in this case, unless such concentration is offset by anæmia, for there is no increase of corpuscular elements, the ratio remaining unchanged.

Clinically the case suggests the following points: From the changes observed in the fundus I would locate the obstruction laterally in the central artery near or at the bifurcation and passing into the lower branch. If the lower arterial system alone were involved, there should be no changes in the upper vessels other than those brought about by doing extra duty or carrying more blood. But they were changed in size, contour, and light streak. An embolus to produce this condition would have to be of that rare variety in which the plug straddles the bifurcation. That the macular region was not more affected is attributed to the peculiar arrangement of the vessels described above. The more normal condition of the vessels at the periphery, and the subsequent return of some of them to their normal calibre are changes described in many of the reported cases of obstruction, and in the absence of any opportunity for collateral circulation in this locality have been thought to be due to backward flow from loss of *vis a tergo*. The keratitis and vitreous opacities are inflammatory, natural results of this condition. The white area so characteristic in these cases, is pathologically infarction, and not œdema, as invariably called. Secondary œdema does occur, but I am told reaches only to the edge of the infarcted area. The process is one of starvation.

TREATMENT. For the general treatment of cases of retinal artery obstruction, it is *important* to determine, if possible, whether or not the obstruction is thrombosis. Against embolism we are helpless, and the other eye will suffer or not, as fate ordains. But if the conditions contributory to thrombosis, as above outlined, can be established, they should be treated until they disappear.

Local treatment to be of any service must be immediate, and that these cases are sometimes seen early is shown by one of embolism

which Dr. de Schweinitz had under observation twenty minutes after the attack.

Now, as to the immediate treatment of arterial obstruction in the eye, whether from embolism or from thrombosis, I am convinced that pressure massage, which has been strongly advised by most writers, is irrational and should not be practised. Pressure on the eyeball stops what flow of blood still exists, and produces the most favorable conditions for further agglutination and accretion. It tends to fortify the obstruction. Surely the indication is to increase the force of the circulation without causing vasoconstriction or to increase the heart action and dilate the peripheral circulation. The drugs of one class will do this, viz., the *nitrites*, and best among these are nitroglycerin and nitrite of amyl. These drugs cause rapid heart action and a dilated peripheral circulation. Gifford has recommended nitrite of amyl. The use of any vasomotor constrictor, which will cause the artery to contract and thus hold tighter the thrombus or embolus, is clearly out of place.

But better than any drug, it seems to me, would be forced muscular exertion, such as running or climbing stairs. In this way the heart could be accelerated, and the peripheral circulation, if affected at all, dilated. Had my patient been enough frightened to run home, I believe she would have been benefited.

Another therapeutic procedure which might be indicated in some cases, and probably was in this case, is hypodermoclysis, or the injection of large quantities of fluid into the cellular tissue of the body for the purpose of rapid absorption into the circulation. Likewise the intravenous injection of saline solution should constitute a part of our resources.

The prognosis of these cases is well known, and I have nothing to add.

EMPHYEMA OF FRONTAL SINUS, FOLLOWED BY EXTRADURAL ABSCESS AND ABSCESS OF FRONTAL LOBE; OPERATION AND DEATH FROM HYPOSTATIC CONGESTION OF THE LUNGS.

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ABSCESS of the brain resulting from empyema of the frontal sinus is a sufficiently rare condition to demand the report of cases. The case which I here present is that of Mr. W. D. L., a boilermaker, aged about forty years, who came to the office of Dr. M. L. Wood, September 8, 1903. For the past six months or more he has suffered from violent headaches, which have steadily increased in severity.